IDIOPATHIC INTERNAL HYDROCEPHALUS (SEROUS MENINGITIS) IN THE ADULT, WITH REPORTS OF THREE CASES (TWO WITH AUTOPSIES).*

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FOR our clinical knowledge of serous meningitis, or primary idiopathic hydrocephalus, in adults, we are chiefly indebted to Quincke. Before Quincke's monograph[1] several cases had been reported by different writers (Huegenin, Eichorst,[2] Oppenheim, Annuske), and the later text-books contain brief references to, or descriptions of, the affection. Eichorst's paper was a valuable contribution, but all the later accounts are for the most part based on Quincke's original study. Most are very meagre and insufficient, and give a very inexact picture of the disease. Oppenheim, however, has given in his text-book a very clear account of the affection. In a later paper[3] Quincke has reported ten additional cases, and makes a further contribution to the subject. But even Quincke's description will probably have to be modified in the future, as many of his cases never came to autopsy, and therefore the diagnosis in these cases must be open to suspicion. Post-mortem findings have been obtained, however, in enough cases to establish the disease and its chief clinical manifestations.

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[1] Volkmann's Sammlung, 1893, No. 67.

[2] Zeitschrift f. lin. Med., 1891, 19, p. 181.

[3] Deutsche Zeitschrift f. Nervenheilkunde, 1896, ix, p. 149.

The pathology of the disease must still, however, be regarded as obscure, and invites further investigation. That the disease process is to be regarded as an inflammation, seems questionable in light of the findings in many of the cases. In one of my cases, for example, even a microscopical examination failed to reveal what are usually regarded as evidences of inflammation. The affection is probably of frequent occurrence, and in the future is certain to be more often recognized after we have learned to be familiar with its clinical features.

On October 12th last, I was asked by Dr. J. C. Munro to see a patient in the surgical wards of the City Hospital. The patient, a woman, had been admitted to the hospital about four weeks previously on account of injury to the head. She had been kicked about the face and head by her husband. This was about four o'clock P.M., and she was said to have been unconscious until about midnight of that day. The next day (September 15th) she was admitted. On entrance the records state "much swelling of left face; whole left face bruised and ecchymosed; some ecchymosis on left side of neck; slight abrasion of left arm and chest; both eyes blackened; marked tenderness over face and entire head; no sign of paralysis." T.: 100.

During the following four weeks (until October 11th) she had exhibited mostly symptoms of a puzzling character; among them irregular but high fever, mild chills, severe headache, obstinate vomiting and pain in the back of the neck were prominent, but not continuous or coexisting. At first there had been delirium, but later her mind seems to have been clear, until the fourth week. Unfortunately the hospital records are not as full as could be wished, in view of the future events, but a study of them with the additional information derived from those in attendance on the case shows the conditions to have been substantially as follows: The above general symptoms were most marked during the first six days; after that improvement set in, and during the following three weeks her condition was fairly comfortable, and, excepting that the fever continued, and at one time one symptom was prominent and at another time another, she might have been thought to be convalescing; but there was a steady and gradual failure of strength. Headache and vomiting were present at times, as well as pain in the back of the neck.

Dr. Bullard saw the patient the day after entrance, and found no objective symptoms of injury to the brain; tenderness of the head and face, headache and vomiting were the most prominent symptoms. T.: A.M. 104.2; P.M. 104.5.

September 17th. (Third day.) "Temperature has dropped about three degrees. Patient complains chiefly of headache and also of general pain. The vomiting continues. Was quite noisy and delirious last night. Seems mentally clear to-day." T.: A.M. 102; P.M. 100.8.

Fourth day. "Patient improving, for the temperature is dropping. The headache is less severe, and though there is still some vomiting, patient says she feels better." T.: A.M. 98.6; P.M. 99.8.

Fifth day. "Patient again shows a high temperature, but the subjective and objective symptoms are no worse. Still complains of headache. Vomits quite often." T.: A.M. 104.5; P.M. 104.5.

Seventh day. The headache and vomiting have subsided, but the patient complains chiefly of *pain* in upper back and in the *back part of neck*. "Diarrhea for the past two days." Temperature still up, 105°.

On the 26th (eleventh day), all the objective symptoms had ceased. Dr. Bullard, who again saw the patient, was unable to find evidences of cerebral disease, and suggested, in view of the exacerbations of temperature and chills, the possibility of malaria, and advised quinine. (This gives an idea of the negative character of the symptoms at this time.)

Three days later Dr. V. Y. Bowditch saw the patient, but was unable to make a diagnosis. Mental symptoms had again returned, but during the following thirteen days the patient continued fairly well so far as local symptoms were concerned, though in general strength she was going downhill. At one time pain in the back of the neck, at another time headache, at another vomiting, were prominent. During the latter week of this period she became noisy at night. It was now learned that for some time before entrance to hospital her actions had been peculiar. She had "had freaks of leaving home suddenly without apparent reason, and returning after some time." At the end of the fourth week (October 11th) local nervous symptoms of importance were noted for the first time, as follows: "Condition has not changed materially until to-day. This morning right pupil was found to be dilated and the left contracted. Patient had a convulsion of the right arm, which began in the thumb and ran upward. Had some twitching of the left arm also. Continued to have twitchings in both arms all day. Some twitchings of the right side of face. Complains of severe pains in back of neck. For the past week has been noisy at night; seems rather dull and stupid to-day."

October 12th. "Slight twitchings of both arms and of the right face continue. Right pupil *less* dilated. Still complains of pain in the back of neck." In the afternoon of this day I first saw the patient. She was dull and apathetic; so much so that when testing the ocular muscles it was with some difficulty that I could induce her to follow the finger with her eyes, or that I could examine the field of vision. The right pupil was dilated and stable. Left pupil slightly dilated and stable (third nerve). Paresis of the right external rectus (sixth nerve).

"There is marked rigidity of the muscles of the back of the neck as well as tenderness of this region. Any attempt to move the head causes considerable pain about the upper cervical vertebræ. Pressure over the vertebræ is likewise painful. She lies with a pad under the neck for relief. Protrudes tongue straight." (I thought there was slight paresis of the right lower facial muscles, but, as this was not observed the next day, I thought this must have been an erroneous observation. In the light of future events I now think this observation was probably correct.) No anesthesia. Nothing remarkable about reflexes. No paralysis of the arms nor legs, but the arms seem to show more resistance to passive motion than normal; a sort of spastic or rigid condition by which attempts to suddenly flex and extend the joints induce contraction of the muscles. This is not marked, but it is unmistakable.

October 13th. House officer reports that in early morning patient mentally much more dull, etc. To my surprise, at visit (about 11 A.M.) patient is comparatively bright; mind fairly clear; laughs; apparently natural, but speaks of an imaginary visit which she says she has made while in the hospital. When asked if she really made the visit or if she dreamed it, she considers for a moment and then says that she dreamed it. Memory fair, but does not remember origin of illness, etc. Still mind not normal. Temperature normal. Abdomen retracted and firm. The paretic condition of ocular muscles is more marked. Right internal rectus is paretic, and the eye does not move downwards as well as the left. Field of vision tested by finger, normal.

October 14th. Change again for the worse; moderate rise of temperature. Mind much more dull and stupid, even than on the 12th. Ocular paralysis more marked still. "Ptosis, right eye. General condition worse, evidently failing. (*No optic neuritis.* Media and fundi occulorum normal (Dr. Bossidy). Examination of ears by Dr. Leland revealed nothing abnormal.)" It seemed evident that the patient was going to die.

On analyzing the symptoms of this case for the purposes of diagnosis it was evident that we had to do with a cerebral disease which appeared to be the consequence of injury. The symptoms were local and general.

The general symptoms which had been noticeable during the course of the disease were: irregular fever, slight chills, headache, vomiting, stupor, delirium.

The local symptoms were: 1, cervical rigidity, tenderness and pain; 2, paralysis of the right sixth nerve; 3, paralysis of right third nerve; 4, paralysis of both pupils; 5, retracted abdomen; 6, mild rigidity of both biceps (arms); 7, localized spasms; 8, temporary weakness of right seventh nerve.

It is noticeable that the local symptoms were latent; that is, that they did not develop until the fourth week of the illness, and that up to that time there was nothing that directed special attention to the brain as the seat of the trouble aside from the fact of the injury to the head.

The general symptoms also showed a peculiarity that should attract attention, although I did not give the weight to it that I should have done, viz., there was a great variability in their intensity (at one time one symptom being the most prominent, at another time another), while the general condition of the patient, with the exception of the temperature, showed a tendency to improvement, followed by relapse. This variability was marked even during the final relapse, after the local symptoms have developed, when the patient was comatose or delirious at one time, and clear in mind at another. The temperature was very irregular, being at times as high as 103°-105°, at other times normal. The temperature taken every four hours showed a similar irregularity.

In making the diagnosis that I did, I was led by the following considerations and reasoning; the history indicated that we had to do with a traumatic affection; the general symptoms (chills, fever, headache, vomiting, delirium, stupor), looked at *aside from their course*, were compatible with meningitis, sinus thrombosis, abscess, and, possibly, with cerebral laceration; the local symptoms (paralysis, spasms, cervical rigidity, etc.), by themselves, were strongly indicative of meningitis of the base.

But it seemed extremely improbable that such a meningitis, presumably purulent, should continue for four weeks without producing local symptoms; in fact, should run a latent course, during which a general improvement should occur, and then end with a sudden exacerbation and development of paralytic symptoms of the basal nerves.

Sinus thrombosis could be excluded by the absence of middle ear disease, injury to the skull, and all evidence of septic infection.

Cerebral laceration was incompatible with the course of the disease.

On the other hand, it seemed to me that the symptoms were compatible with, and explainable by, abscess of the brain. Trauma is known to cause abscess, which also may run a latent course. It seemed to me at the time, that the general symptoms were explicable by a latent abscess, which finally, by pressure, or possibly by rupturing and thereby exciting a meningitis of the base, had caused the local symptoms. The strongest point against this theory was the temperature, which in latent abscess is said to be usually normal or sub-normal; but a study of recorded cases showed that this statement is too general and that fever is not uncommon. Besides, in fact, fever is also uncommon in acute hydrocephalus or serous meningitis.

For reasons unnecessary now to go into, the most likely location of such an abscess seemed to be, first, the cerebellum, and next, the tempero-sphenoidal lobe. Accordingly, while recognizing the uncertainty of the diagnosis, I felt justified in advising an exploratory opening. On the 25th, Dr. Munro operated, exploring first the cerebellum, and then the base, with negative results. Not finding any pus in the cerebellum or about the base, it was decided to make a second opening over the tempero-sphenoidal lobe, but before this was completed it became evident that the patient was bearing the operation badly, and it was thought best not to go farther.

The patient only partially regained consciousness after the operation, and continued to fail. Death took place on the early morning of the 18th, the third day after the operation.

Autopsy, Oct. 19th, 1896, 32 hours post-mortem, by Dr. F. W. Draper.

The body was that of a woman 29 years old, greatly emaciated.

There were scarcely visible remains of ecchymoses in the left temporal region, without definite outlines, and yellowish in color, with a bluish tinge.

The left pupil was larger than the right.

The left temporal muscle showed a slight degree of infiltration with blood.

The skull was not fractured and showed no injury whatever, except the recent surgical lesions.

The dura was normal.

The surface of the brain at the vertex showed complete obliteration of the sulci and flattening of the convolutions, with injection of the vessels of the pia; the cerebral face was dry and sticky, but not inflamed.

On palpation of the brain laterally there was abnormal elasticity.

At the base there was more than the normal amount of cerebro-spinal fluid, with a moist and sodden state of the brain tissues exposed.

The vessels at the base were normal.

There was no pus in the arachnoid spaces.

Section through both of the lateral ventricles found those cavities dilated to about three times their normal capacity; they contained clear fluid with a yellowish tinge to the amount of five fluid ounces.

The ependyma was pale, slightly swollen, velvety, showing a sodden aspect.

The velum interpositum was pale, swollen and moist.

There was no appearance of injury, old or recent, on section of the cerebrum.

The brain substance was moist, but not injected.

The heart, lungs, spleen, stomach, pancreas, intestines, liver, gall-bladder, kidneys, supra-renal bodies, great vessels, womb, bladder and ovaries were normal.

Case II.—C. G., housewife, aged 30. Previous history given by Dr. R. C. Macdonald, under whose constant care she was from August, 1888. Well and strong until second confinement, in 1891; never well since then. A pronounced neurasthenic "of that unsatisfactory kind that nothing seems to help;" sometimes better, sometimes worse. Chief suffering of menstrual periods; that is, attacks of prostration, vomiting, headache, abdominal pain and great tenderness, extreme wakefulness and nervousness. Sometimes slight chills and slight fever; no stupor nor delirium. Between attacks, fairly well. Attacks grew progressively worse, and lasted about two weeks. During last year vomiting was a prominent symptom. Neurasthenic and other symptoms were thought to be due to ovarian disease, and ovariotomy advised. (At the autopsy cysts were found.) Dec. 10th, 1896, well, excepting for a cold. December 19th, one of her menstrual attacks.

December 26th, headache began; said to have been quite well since December 19th. On December 29th reported better, excepting for severe headache, but not seen until January 2d; then chills; temperature, 103; headache, vomiting, etc. Diagnosis: Suppressed menstruation, peritonitis, subacute and general inflammation of the pelvic organs. Admitted to Boston City Hospital, Jan. 5, 1897; service of Dr. A. L. Mason. Patient complained, previous to admission, of chills of an indefinite intensity and character. Condition on entrance: Complains of *pains* all over, and especially in the head and neck; frontal and occipital headache; also an area of pain along the sagittal suture; neck is very sore, and movement of the head causes pain. Some, but not localized, pain in the abdomen, also in genitalia and across chest. Physical examination: Organs negative; knee-jerks much diminished; no ankle clonus; no edema nor anesthesia; *tenderness of the muscles of the neck, with tendency to retraction.* Temperature, 100; pulse, 80.

Continuation of this condition for about twelve days. Examination of the blood for malarial organism, negative. *Slight optic neuritis;* no otitis. During the first two weeks after admission the temperature was very irregular, the highest point reached being 103½, while at times, for short intervals, it was normal. After the first two weeks, temperature was normal or subnormal. Urine at first normal, but at one time later contained one-half per cent. albumin, blood and casts. Thirteenth day after admission, change for the worse; comatose; involuntary movements of the bowels and bladder. This condition persisted during the following six days, when Klebs-Lœffler bacillus was found in the throat without inflammation. Patient removed to the contagious wards (South Department) to care of Dr. McCollom.

Records of this department state condition on admission as follows: Pulse slow, regular, good volume and tension; pupils equal and react normally; no retraction of the head; slight tenderness over nape of neck; intelligence stupid; heart, lungs and throat negative; decided muscular weakness. For a short time after admission patient was obliged to be restrained to be kept from falling out of bed, which she did once or twice. Temperature slightly subnormal during first three weeks, after that normal. Pulse varied between 60 and 90 degrees.

Ten days later it was noted that the facial expression was that of a dement. Facial muscles smoothed out; pupils became dilated and reacted sluggishly to light and accommodation. During the following four weeks patient remained in the same condition of dementia. The coma had passed away, but the intelligence varied; sometimes the patient seemed brighter, and at other times more demented; occasionally some delirium. A good deal of nausea and vomiting, and more or less headache. But excepting condition of the pupils, no focal symptoms; no paralysis, nor anesthesia, nor tremor, but general muscular weakness. Speech, muscular co-ordination and reflexes normal. On several occasions the house officer was called at night because of the poor condition of the pulse; in a few minutes it would become of fair strength; these spells similar to the one before her death.

March 4th. No special change, save that for the last few days has complained of constant frontal headache; says, "I believe my head will burst." Some vomiting. Temperature subnormal for the past six days; pulse about 60. In the afternoon an attack in which the pulse became several times alternately almost imperceptible and of fair strength. Half an hour later, death. Duration of illness about eleven weeks.

From the above it will be seen that the prominent symptoms in this case were: Impairment of consciousness, viz., delirium, stupor, coma and dementia; the last simulating, as was thought at one time, general paralysis. Chills and fever at first, later normal and subnormal temperature; rapid, followed by slow pulse; abdominal pain and tenderness, and cervical pains; severe headache; nausea and vomiting; optic neuritis; muscular weakness, but no paralysis noranesthesia. Some of these symptoms appeared and disappeared during the course of the disease, which exhibited temporary improvement with relapses. The demented facial expression, a part of the general muscular weakness, and the absence of spasms, are worth noting. The different diagnoses of different attending physicians give an inkling into the general picture of the disease at different times: "Peritonitis," "Cerebro-spinal Meningitis," "General Paralysis."

Autopsy: Kidneys, weight 300 gms. Very firm, of a dark-red color. Cortex injected. Relation of cortex and pyramids normal.

Both ovaries cystic, cysts varying in size from a small pea to that of a walnut. Bilateral laceration of cervix

Brain: weight, 1095 gms., small. Convolutions flattened markedly over convexity, particularly over frontal lobes; in places practically abolished. No areas of softening. The pia strips easily, leaving a moist, soft, smooth surface. The gray matter has less color than normal. Brain substance easily torn, clings to knife. The punctæ cruentæ absent. Ventricles contained about 150+cc. of clear serous liquid. Ventricles markedly dilated, particularly the fourth ventricle. Ependyma smooth, shiny, no adhesions nor roughenings anywhere. Brain markings normal, except that basal ganglia are paler than normal. Vessels at base show no sclerotic changes. Cerebellum and medulla moist, otherwise normal.

Cervical and beginning dorsal cord soft, flattened out as cord is laid across finger. Markings less distinct than normal. Other organs not remarkable.

Microscopical examination of the ependyma, made by Dr. J. J. Thomas, showed "no changes beyond a slight increase of the neuroglia lying beneath the ependyma, and no evidence of any inflammatory changes. There is some infiltration about the blood vessels of the brain, and signs of atrophy of the brain."

Anatomical diagnosis — Hydrencephalus with edema of the brain. Hypostatic congestion of lungs and kidneys. Slight arterio-sclerosis. Cystic degeneration of ovaries; bilateral laceration of cervix.

Case III.—The following case, which I saw in consultation with Dr. Henry F. Leonard, probably belongs here, although, as recovery took place, the diagnosis must remain unconfirmed, and rest on exclusion. Mrs. B., middle age, dressmaker. Condition at time of consultation: Semi-comatose; aroused with difficulty; temperature normal or subnormal; no paralysis; incontinence of bladder and bowels; urine normal; heart negative; no pulmonary symptoms; pulse weak. Patient seemed moribund, and an absolutely unfavorable prognosis was given. The history, which it should be said Dr. Leonard has given me from memory, is as follows: *Onset*—well until the day she was taken ill; worked during forenoon, but severe headache, for which she consulted Dr. Leonard. Besides headache, felt as if she was falling backwards; "eyes were blurred at time of first visit." *Progress*—Headache continued; no vomiting at first, later several attacks; pain in almost every part of the body; screamed when touched or when moved; temperature normal at first, later subnormal; no paralysis, anesthesia nor spasms; urine normal; no cough nor pulmonary symptoms; delirium; muttered much at first, talked to imaginary persons, then coma or stupor; more or less unconscious for three weeks or longer; gradual loss of flesh and strength each day; "ice-cold" extremities; dilated pupils; low respiration; slow pulse; nourished by rectum. "For about two weeks seemed to be on verge of collapse." "During the illness, and for several months after the acute symptoms had subsided, she had a vacant, idiotic expression" (facial paresis?). Gradual recovery. *Duration*—Two months. Fully six months before headache was gone—much longer time before she could work. Eyes so weak that she could not use them for several months.

No other diagnosis than serous meningitis seems possible in this case. The disease was plainly cerebral, and if cerebral, probably a meningitis; the absence of fever, the prolonged course, complete recovery, absence of palsies, render the purulent form unlikely, and point strongly to the serous variety.

In view of the probably frequent occurrence of the affection, its importance, and the very brief references or descriptions that are to be found in English literature, a very short résumé of our knowledge may prove acceptable. The following account is for the most part based on Quincke's monographs:

The fundamental conception pertaining to the affection it that a meningitis, particularly an ependymitis, may give rise to a simple serous effusion, as well as a purulent one; just as an inflammation of the pleura may cause serous effusion into the pleural cavities, and one of the synovial membranes an effusion into the joints. Thus we may speak of a serous meningitis or a purulent meningitis. Hitherto it has been customary to picture meningitis only in its purulent (or tubercular) form. By bringing meningitis into analogy with inflammation of other membranes (pleuritis, pericarditis) we enlarge our conception of the disease.

As such a meningitis may occur at all ages, and give rise to collections of fluid in the ventricles, we have a primary idiopathic internal hydrocephalus of adults, as well as the classical form of children.

The causes, so far as known, may be regarded as trauma, mental strain, alcoholism, otitis media and acute infectious diseases.

The affection may be either acute or chronic.

The acute form in its symptoms simulates purulent and tubercular meningitis.

The chronic form presents symptoms so closely resembling tumor that it is usually diagnosed as such. According to Quincke, in another variety of the chronic form the symptoms are those of neurasthenia. It may be pointed out, however, that as the conclusive proof of an autopsy is necessarily lacking in such cases, the existence of this neurasthenic form must be open to doubt, and must rest on theoretical considerations. The previous neurasthenic condition in case II. above is strongly suggestive of this view.

The affection is not so rare as would appear from the paucity of reported cases, the acute cases being mistaken for purulent meningitis, and the chronic ones for tumor. Even when at the autopsy an effusion is found it is not customarily regarded as pathologically a sufficient cause of death.

The cases may be further classified as follows:

I. Acute onset.
(a) With acute course.
(b) With chronic course.
II. Chronic course.

(a) With chronic progressive course.

(b) With acute exacerbations.

The symptoms, as in other brain affections, are general and local, and depend largely upon increase of pressure. The general symptoms are headache, vomiting, fever, somnolence or stupor, delirium, convulsions, optic neuritis. The local symptoms are paralysis (especially of the cranial nerves), exophthalmos, cervical pain and rigidity, hyperesthesia, pain in extremities, etc. It is by its course, and greater or less intensity of individual symptoms, that it is to be distinguished from other forms of meningitis and tumor.

When the onset is acute it may resemble purulent meningitis, but more often it is slower as in the tubercular form; but even the latter, in adults, is more rapid and more continuously progressive. Nevertheless, there are cases which are apoplectiform in their onset, with loss of consciousness. The pulse is often slow. Fever is either absent or only slight, of short duration and irregular course. (To this, Case I., above reported, is an exception.) The headache is diffuse, or located in forehead or occiput, of varying intensity and sometimes periodic, or with periodic exacerbations. Usually, some days after the onset, dulness of consciousness is observed. As a rule, this is manifested later than in purulent meningitis. It is only in fatal cases that this is continuous and profound. Usually, like headache, it is of varying intensity and alternates with, or is associated with, unrest, delirium and sleeplessness. Vomiting is common. Rigidity of the neck, with tenderness and pain of motion, may be prominent. (This was marked in Cases I. and II.) Paralysis of the cranial, particularly ocular and facial, nerves may be present, though not usual; that of the sixth is most common, it being most subject to pressure on account of its course. These paralyses are apt to be slight and of varying intensity. The pupils are unequal and react slowly, or are stable. Oppenheim describes weakness of the extremities as common.

Spasms, more or less diffuse, may occur. An important finding is optic neuritis, with atrophy. This is more frequent than in other forms of meningitis. In the chronic cases it is frequently associated with headache, vomiting and mental dulness as the cardinal symptoms, thus simulating tumor; but after weeks or months such cases may end in recovery. The visual defect, instead of blindness, may be that of bi-temporal hemianopsia due to pressure from the dilated third ventricle upon the optic chiasm.

Other symptoms are exophthalmos, hyperesthesia, pains and intention tremor. In Case I. rigidity of the abdomen and arms was present. A demented facial expression was noted in two of the above cases.

The acute cases may, after a course of some weeks, end in complete or incomplete recovery or death, or become chronic. A remaining optic atrophy may be the sole indication of past cerebral disease. The chronic cases may pursue a varying course, with ups and downs, and acute exacerbations. Such an exacerbation may, in the absence of a complete history, give the erroneous idea of an original acute attack. (In Case I. it is a matter of doubt whether the trauma caused such an exacerbation or an original attack.) They may continue for years, ending finally in recovery or in death. For the diagnosis, which at present in most cases must be extremely difficult, Quincke lays stress upon the great variations in the intensity of the symptoms from day to day; at one time one symptom, at another time another coming to the foreground. In the chronic cases the occurrence of remissions and intermissions must largely be relied upon to distinguish them from tumor, and when the not very common focal symptoms (palsies, etc.) are present, the fact that instead of progressively deepening, as with tumor, these things have a temporary or varying existence.

The pathological findings are for the most part limited to the accumulation in the ventricles of clear fluid which does not materially differ from normal. Quincke makes the point that while the effusion into the arachnoid from cortical meningitis is almost always turbid, and therefore more or less rich in cellular elements and albumin, the reverse is the case in meningitis of the ventricles.

The ventricles may be enormously distended, and the pressure so increased that the convolutions may be flattened and the sulci obliterated in appearance. The changes in the ependyma seem to be limited to hyperemia, in the acute stages, and to some slight thickening and change in texture (described as smooth, velvety, granular, sodden) in more chronic stages. The pia may be hyperemic also, and share somewhat in the process. Quincke, in discussing the pathogenesis, likens the affection to angio-neurotic acute edema of the skin, an analogy which renders intelligible the sudden development and variability of tension (symptoms) observed.

It is not quite clear to my mind that the effusion is to be regarded as of an inflammatory character. Certainly in Case II. above, the condition of the ependyma does not suggest inflammation, and the same may be said of the findings in some of Quincke's cases. The pathology of the effusion seems to me to be obscure and deserving of further research.

As to treatment, Quincke ascribes recoveries to the use of mercury. Lumbar puncture has its advocates. In cases which pursue an unfavorable course and a fatal issue seems imminent, I should be inclined to advise tapping the ventricles, although much might be argued against this procedure on the ground that its effect would be likely to be only temporary.

Case IV.—The following case is of interest in this connection, although the cerebral condition was complicated by, or caused by, a sunstroke, and the nature of the meningeal effusion may be questioned. At the autopsy chronic ependymitis was found, but the serous effusion was confined to the convexity of the brain. If this effusion can be properly regarded as that of a meningitis and not edema, the case is an example of the serous form of external meningitis.

J. D., age 15, school-boy. Previous history—Boy has always been mentally backward (probably from not having gone to school until 12 years old), but physically well.

Present illness—Had been playing out-of-doors in the sun all day—the day of entrance; in afternoon was sent on an errand; later discovered on a doorstep in an unconscious condition. Admitted to hospital, service of Dr. Jackson. Condition on entrance—Unconscious; head retracted, cyanotic; hot, dry skin; breathing rapid and noisy. Eyes rotated upward and to left; pupils pin-point and stable; conjunctivæ injected; pulse regular, rapid, of poor strength and volume; temperature 110; arms slightly flexed and rigid; legs negative; reflexes present; other organs normal. Treated with ice-cold sponging and packs. Temperature reduced at first; later rose; irregular; fell to normal on the fifth day, then rose and continued between 102 and 103. Profuse vomiting and involuntary ejections. Unconsciousness continued for three days. Temperature required several cold packs each day; nourishment taken; marked rigidity of the neck, trunk and extremities.

Fifth day—Patient seemed conscious, though stupid. Will answer a question by yes or no. Temperature still up. Pulse fairly strong. Three sponge-baths given to-day.

Ninth day—Pupils small, left being somewhat larger than right; neck rigid; constant convulsive movements of extremities; occasional twitching of face; puts tongue out when spoken to; takes a little milk with a spoon; keeps muttering a good deal; constant vigil; passes urine involuntarily; bowels constipated lately. Dr. Lancaster found "no neuritis or other abnormality of fundus."

Thirteenth day — Patient's condition is decidedly worse. There is now profound unconsciousness, with less convulsive movements of hands; respiration rapid; temperature between 101 and 103; pulse weak and rapid; takes very little nourishment; loose movements of bowels to-night after repeated cathartics.

Fourteenth day—Patient failed steadily all day. The pulse grew weaker and weaker, and the respiration became more shallow, until he died at 11.55 P.M.

Autopsy—Brain: pia extremely distended by a large amount of slightly thickened, cloudy serous fluid. The brain looked as if covered with a thick layer of wine jelly. Vessels of pia markedly injected. The right lateral ventricle had noticeable adhesions between opposite layers of the ependyma; velum interpositum thickened and contracted; no edema of any organs of the body; other organs, with the exception of emphysema of lungs, negative.

Case V.—Under the title of "A Case of Recurrent Basilar Meningitis," Dr. Henry M. Koles reports[4] a case which, it seems to me, in all probability, was of the serous variety, with effusion into the ventricles. The patient, 23 years old, had previously suffered from several (four) similar attacks of varying, but, as compared with the last attack, less severity. The first followed a boxing bout, when he was thrown against the edge of a table and rendered unconscious. By one attack there was paralysis of one arm and leg. At one period he suffered from attacks of headache, recurring every six months, preceded by vomiting, and confining him in bed for several weeks at a time.

Final attack—Onset sudden; vomiting, headache, restlessness; fever (T. 101), rapid full pulse. Recovery in the course of a few days; return to business followed, after a few hours of the same day, by sudden faintness, dizziness, and nausea; severe vomiting. Again fever, rapid pulse, flushed and anxious face, labored breathing; retraction of the head. During following ten days progressively worse, various paralytic symptoms developing one after another. Symptoms — Pain in head, abdomen and back of neck, and rigidity of muscles of neck. Dilated and stable pupils, exophthalmos, then ptosis and double sixth-nerve paralysis. Neck rigid and immovable. Increased pain in head, and pain down the arm. Restlessness and delirium; maniacal. Left facial paralysis. Highest temperature, 103;

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[4] Med. Rec., Oct. 10, 1896.

pulse, 140. Optic neuritis. Apathetic and delirious by turns. Cheyne-Stokes respiration. Death apparently impending, but instead unexpected gradual improvement and recovery. Treatment anti-syphilitic, though no history of syphilis.

The exophthalmos, double sixth-nerve paralysis, and dilated pupils, are plainly indicative of increased pressure from effusion into the ventricles.

It has already been stated that the chronic cases simulate tumor, and, according to Quincke and Oppenheim, are usually diagnosticated as such. It is highly probable that the "two cases in which signs of the presence of an intracranial tumor were followed by recovery," lately reported by Dr. J. Mitchell Clarke,[5] were of this nature; and if this interpretation of these interesting cases is correct, they may be quoted here in corroboration of this statement. In both cases there were optic neuritis, intense headache, vomiting of nervous origin and vertigo. In the second case there were, in addition, chills, muscular twitchings and cramps, and pains in the occiput and jaw. Both pupils were dilated, the left being greater than the right, and both reacted sluggishly. There were drowsiness, mental irritability and delirium. Clonic spasms of one hand were also noted. In the first case there were sudden attacks of blindness, lasting two or three minutes. The temperature in both cases was normal or subnormal. The duration of the first case was eight months, of the second, four months. In the first case there were evidently periods of improvement followed by relapses, as the "patient returned to work at irregular intervals." Both cases recovered, but with persisting blindness. The theory of the reporter is that they were cases of tubercular tumor which had become encapsulated, but he does not seem to have taken into consideration the possibility of "serous meningitis," although the common form of meningitis, and other possible conditions, were considered in making the diagnosis.

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[5] British Medical Journal, Feb. 6, 1897.

DISCUSSION.

Dr. Philip Coombs Knapp, of Boston—I suppose that Dr. Prince has spoken, in the part of his paper which he omitted to read, of the importance of lumbar puncture in the diagnosis between this condition and the tubercular form of meningitis. I simply bring this forward now because the last two or three months in Boston have shown very clearly the value of the lumbar puncture in the diagnosis. There has been an epidemic there, some thirty cases having come to the hospital in the last few months, and lumbar puncture has been practiced in every case, with very valuable results, differentiating clearly between the epidemic cerebrospinal form and cases, which presented similar clinical pictures, of tubercular meningitis. The negative findings after lumbar puncture seem to me most valuable in determining between this so-called serous meningitis and the other forms.

Dr. Joseph Collins, of New York—There can scarcely be any doubt that this condition occurs. As to whether or not the process is an inflammatory one depends entirely upon the meaning we put upon the term inflammatory. If we incline toward the view that no condition is inflammatory unless it is attended by a vascular exudate, then the morbid process at the bottom of this condition is not an inflammatory one. If, however, cellular proliferation is an indication of inflammation, we must admit that there are cell changes which go on in the ependyma, *i.e.*, in the internal lining of the ventricles, and, indeed in the pia, that must be considered inflammatory; in short, there is a reproduction of cells, and new cellular formation, and this new cell formation is commensurate with cell disintegration and progresses, the former endeavoring to supersede the latter.

I am glad to hear Dr. Prince say that in all probability there are chronic forms of this condition. I venture to believe that chronic, or subacute, forms may be of more frequent occurrence than the acute. It is possible that some of the obscure intracranial manifestations occurring in children, which we are now unable to interpret, may be due to this morbid state of the ventricles. I am prompted to this remark because of an experience which I have recently had with a case that conformed to the description of hereditary cerebellar ataxia in almost every particular, except that there were *pied bot* and peculiar trophic muscular changes. The case was demonstrated before the New York Neurological Society, and the diagnosis of hereditary cerebellar ataxia was confirmed. The patient came to autopsy a number of months ago, and a ventricular distension, very like that described by Dr. Prince in one of his cases, was the only departure from normal that could be found to explain the symptoms from which he had suffered for many years. I say *explain* these symptoms, but I do not mean to say that the ventricular condition did truly explain the symptoms. At least the cerebellum was apparently normal to the unaided eye, but this does not preclude the existence of changes in the latter which will be discovered later. The case impressed upon me the importance of thoroughly exhausting the possibilities of ventricular conditions before making a diagnosis of structural disease of the substance of the encephalon.

The President—I would like to ask if there was a microscopic examination of the cerebellar cortex in the case to which you refer.

Dr. Collins—No; the microscopic examination has not yet been made.

Dr. Sachs—I think we ought to hold this matter in abeyance; I do not think that Dr. Collins would be willing to say that the ventricular distention which he found in that case had any direct bearing; and until the examination is entirely complete, I do not think it would be wise to have our views distorted by any findings of that sort. I think we must not be too hasty to force a single pathological finding to answer as an explanation for an hereditary disease which has as distinct symptoms as hereditary cerebellar ataxia.

Dr. Morton Prince—Undoubtedly there is a chronic form of this affection, and it is probably just as common as the acute form, but the chronic form generally simulates, and is usually mistaken for tumor. There is present optic neuritis, headache, vomiting, etc.. The principal differential point in the diagnosis is the variation in the intensity and persistence of the individual symptoms. While in tumor the symptoms are fairly constant, in the other condition they may vary largely in their intensity from time to time. The chronic form may be chronic from the beginning, or it may be the outcome of an acute case. The acute cases may get well, or may become chronic cases.

There is also another chronic form which it is claimed exists, at least by Quincke, and that is the neurasthenic form. He claims that in certain cases the symptoms are of a neurasthenic character. This is difficult to prove, because naturally such cases do not come to autopsy; but I would call attention to the second case which I reported, which had been a neurasthenic for a very long period before the final attack, and in this neurasthenic condition she had attacks very similar to the attack which finally ended her life. That case seems to me to be of value as evidence in favor of the statement that the symptoms may be neurasthenic in the chronic form. I am inclined to think this condition may be far more frequent than is generally supposed.